

Some Aspects of Parietal Lobe Disorders.

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Despite an extensive literature on "parietal lobe syndromes", the impression appears still to exist that such disorders are comparatively rarely encountered. It therefore seemed of interest to compare the frequency of some of these syndromes with that of uncomplicated hemiplegias. This Thesis is concerned with observations on cases admitted to general medical wards in the Western General Hospital, Edinburgh, under the care of Dr. W.I.Card, during the period October 1948 - September 1951. During this time, thirty-seven uncomplicated hemiplegias and thirteen cases showing such disorders as autotopagnosia, anosognosia for hemiplegia, sensory inattention, various forms of visual-spatial agnosia, and Gerstmann's syndrome, were seen.

In addition, five other patients were admitted, whom in life it was impossible to investigate adequately on account of their comatose condition, but who at autopsy were found to have varying degrees of parietal lobe destruction.

Brief accounts of the thirteen cases above mentioned are now presented, followed by analyses of the main syndromes encountered, and a general discussion of the symptomatology and clinical investigation of parietal lobe disorders.

Case Reports:

1. Mrs.D.E. Aet. 69. Admitted 27.12.48.

History - On 26.12.48, she became giddy, fell down, and was unable to rise. She did not injure herself seriously, did not lose consciousness, and had no headache or convulsions.

Previous History - For twelve years she had suffered from breathlessness on exertion, and for six years from ankle oedema.

Family History - None of relevance.

Social History - A housewife in comfortable circumstances.

Examination - She was an obese and somewhat confused patient, with generalised arteriosclerosis. She was right-handed. She had a left homonymous hemianopia and a left hemiparesis, with involvement of the tongue and palate. The optic discs were normal. The tone and deep reflexes of the limbs on the right side were increased, and the right plantar response was extensor as well as the left. She appreciated crude stimuli on the left side of the body, but displayed alloaesthesia. Sensation in the right side of the body was normal. There was no evidence of pain of thalamic origin. She seemed to be unaware of the left half of the body, and claimed that she was quite well and free from any weakness, thus displaying both autotopagnosia and anosognosia. The delusions persisted when the paralysed limbs were

brought within the compass of the intact half-fields of vision.

The pulse was regular, the blood-pressure was 170/104 mm.of mercury, and the heart was moderately enlarged. The retinae showed second degree hypertensive changes. There was no evidence of cardiac failure. Lumbar puncture was not performed, but the blood Wasserman reaction was negative.

A diagnosis of cerebral thrombosis, involving the right internal capsule and thalamo-parietal projection, was made. The patient was got up early and given physiotherapy, but her condition did not improve, apart from the disappearance of autotopagnosia four days after admission, at which time she also lost her mild confusion. Yet the anosognosia remained. She died from bronchopneumonia on 5.1.49, and unfortunately permission for autopsy was refused.

2. J.D.R. Aet. 68. Admitted 7.6.49.

History - Since 1948, he had suffered from unsteadiness of gait, headaches, and a right homonymous hemianopia. His right hand had grown progressively weaker, and he had become dysphasic, although he had not noticed any difficulty in reading.

Previous History - None relevant was obtained.

Family History - His mother died at the age of forty years from a cerebral vascular accident.

Social History - Retired, in reasonable circumstances.

Examination - A right-handed patient, he had a right homonymous hemianopia. The fundi were normal, and there were no cranial nerve defects. A right hemiparesis was present, with only slight increase in tone and deep reflexes in the affected limbs. The right plantar response was extensor. Marked constructive apraxia was present in the left hand. He had a severe receptive dysphasia of the nominal type, and also palilalia, dyslexia, dysgraphia (in excess of what might be expected to result from his paresis, which was not severe), and object agnosia. He was able to draw, though imperfectly, but defects were not limited to one side of the drawing. Bisection of lines, and marking the centre of a circle, were correctly performed. He could find his way about, and could correctly describe routes. He was able to dress himself, albeit somewhat clumsily.

Sensation was normal, and he had no sensory inattention. There was no finger agnosia, aphasia or apraxia, despite other manifestations of agnosia, dysphasia and apraxia previously mentioned, and discrimination between right and left was intact, both for the patient's own body, and those of other people.

No signs of note were present in the other main clinical systems.

A diagnosis of neoplasm of the left parietal lobe was made, and exploration was carried out in Professor Dott's department in the Royal Infirmary. At craniotomy, a cystic tumour was discovered in

the left parietal region, and deemed inoperable. (Pathological examination of a biopsy specimen confirmed that the growth was a Glioblastoma Multiforme). The patient died on 25.7.49, and permission for further examination was refused.

3. J.McA. Aet. 55. Admitted 19.6.50.

History - This patient was not fully conscious on admission, but a history was obtained that earlier on 19.6.50 he had felt giddy. Loss of consciousness supervened, but by the time of admission he had partly recovered, although he was still drowsy and confused. He had not had any fits or vomiting, and had not injured himself.

Previous History - There was no relevant previous history.

Family History - His mother and an uncle had died from cerebral vascular accidents.

Social History - A butcher in comfortable circumstances.

Examination - The patient was initially confused, and complete examination was therefore not at first possible. It was noted that a left hemiparesis was present. The cranial nerves were normal, apart from the upper motor neurone facial lesion. The fundi were normal. There was no evidence of a fracture of the skull, and the ears were healthy. The pulse was regular, the blood-pressure was 140/90 mm. mercury, and the heart size and sounds were normal. No obvious

source of emboli was discovered.

On the following day, he had recovered sufficiently to be examined more fully. He was found to be right-handed. He had no hemianopia or hemi-hypaesthesia, but well marked visual and tactile inattention were noted, and there was astereognosis in the left hand. There was neither anosognosia nor autotopagnosia, and he had no apraxia (including apraxia for dressing).

X-rays of the skull were normal, the blood Wasserman reaction was negative, and the cerebro-spinal fluid was in all respects normal.

Further testing revealed that this patient had a notable inability to draw maps, either freehand or on copying. The left side of the map would be incompletely drawn, or rendered wholly inaccurately, in contradistinction to the right side, which was usually reproduced tolerably well. When asked to indicate the positions of the principal towns, he would frequently place them in the sea to the right of the land; and this error he continued to make, despite his attention being drawn to his mistakes. East and West were often misrepresented as North and South. (See Figures 1 & 2). This patient was not unintelligent, and the production of maps should normally have been well within his powers. Despite this difficulty, he could copy simple designs with matches in two and three dimensions. He was able to describe routes correctly, and later, when able to get up, he had no difficulty in finding his way about without mistakes.

The hemiparesis disappeared rapidly, but tactile and visual inattention remained for a fortnight. When he was discharged on 5.7.50, his rendering of maps was still defective, though some improvement was evident. He could correctly bisect a line and mark the centre of a circle.

He has been kept under observation since, and has remained symptom-free. Map-drawing has gradually improved.

Some hesitation was felt about the nature and localisation of the cerebral lesion. There was at no time any evidence of a source for emboli or of neoplastic disease outside the skull; and the initial investigations, and the natural history of the case over a period of months did not suggest the presence of a primary cerebral tumour. It was ultimately felt that the patient must have had some spasm of the right middle cerebral artery, with secondary thrombosis of the branches to the parietal cortex. Campbell (personal communication) is of the opinion that some cases at least of cerebral thrombosis start with cerebral arterial spasm.

4. Miss M.W. Aet. 54. Admitted 5.8.50.

History - On 4.8.50 she developed dizziness and difficulty in thinking. She then fell, but did not hurt herself or lose consciousness. She had no convulsions, headache or vomiting. She found that she could not express herself properly, but swallowing was unimpaired. She was admitted to hospital on the next day.

Previous History - She had suffered from breathlessness on exertion for two years, and for some months had complained of headaches.

Family History - None of relevance was obtained.

Social History - She was employed in a responsible position in charge of clerical workers in a printing and publishing business.

Examination - A right-handed patient, she was correctly oriented. She had no evidence of hemiplegia, hemi-hypaesthesia or hemianopia, and no sensory inattention. She suffered from moderately severe dysphasia, mainly receptive, and constantly resorted to periphrasis. Written instructions were at first better understood than spoken ones. Her own powers of writing, whether spontaneously, on dictation, or copying, were initially poor. She also suffered from acalculia, and had difficulty in distinguishing right from left both as regards her own person, and that of her examiner. She showed neither finger agnosia nor finger apraxia, but displayed finger aphasia. Her execution of plans was faulty in the

extreme (See Figure 3), but she did not neglect any particular side - right or left, top or bottom. She could bisect a line and mark the centre of a circle. A mild degree of constructive apraxia was observed.

Her pulse was regular, and the blood-pressure was 210/140 mm. mercury, falling with rest to 168/100 mm. mercury. The heart was moderately enlarged, and the retinae presented Grade 2 hypertensive changes. The urine and blood urea nitrogen were normal. An electro-cardiogram showed changes characteristic of left ventricular strain. The blood Wasserman reaction was negative, and the cerebro-spinal fluid was normal in every respect, and under normal pressure. X-rays of the skull suggested some degree of calcification of the internal carotid arteries, but were otherwise unremarkable.

The patient made quite rapid progress spontaneously and when discharged on 30.8.50, she was able to speak with reasonable fluency, she could read and write with only occasional mistakes, and her execution of plans was satisfactory (See Figure 4).

It was not considered that all the features of her case could be explained on the basis of a parietal lesion, as some of the defects of speech and writing were motor in character. But it seemed reasonable to suppose that she had a lesion, probably due to a thrombosis, in the left parietal cortex in the region of the supramarginal gyrus, extending towards Wernicke's zone. The inability to draw plans

raised the possibility of a lesion in the right parietal lobe as well, but this is problematical, and will be considered in the discussion hereafter.

This patient has been seen periodically since discharge, and has shown only minor residual defects of speech, writing and calculation.

5. W.O. Aet. 68. Admitted 16.8.50.

History - This man's daughter stated that for three days he had suffered from left-sided weakness, and confusion, although he had not lost consciousness. Speech had been impaired as a result of confusion.

Previous History - He had suffered from myxoedema for five years, and angina pectoris for four years. No previous history of neurological symptoms was obtained. The myxoedema had been treated with thyroid, but inadequately.

Family History - None of relevance was given.

Social History - A retired professional man in comfortable circumstances.

Examination - A left-handed patient, he had been made to write in childhood with his right hand. He was moderately myxoedematous (though not sufficiently so to render neurological investigation difficult), and had diffuse arteriosclerosis. He had signs of a left hemiparesis, with hypaesthesia in the left half of the body, but no hemianopia. Although there was no evidence of autotopagnosia or anosognosia, he paid little attention to the left half of extra-

personal space. Visual and tactile inattention were well-marked. Despite his left-handedness, there was no sign of a Gerstmann syndrome.

The pulse was regular, and the blood-pressure was 110/75 mm. mercury. The heart was moderately enlarged, but there was no evidence of pulmonary or systemic congestion. An electro-cardiogram showed changes consistent with coronary disease, but not with myocardial infarction or myxoedema heart disease.

It was considered that the patient suffered from a thrombotic lesion in the right internal capsule and the thalamo-parietal projection, notwithstanding the slight nature of the sensory defect.

He was treated with thyroid, and graduated exercises after an initial period of rest. He made a good recovery. Visual and tactile inattention disappeared after ten days, but neglect of the left half of extra-personal space was evident for six weeks. When he began to walk, he avoided the left half of the ward and consistently turned to the right when altering his direction. On walking out of the ward he would always turn into the corridor to the right, not to the left. Nevertheless, his description of places and journeys was accurate, and his ability to draw freehand and on copying was normal once power returned to the left hand, with no sign of neglect of the left side of the drawing. He could bisect a line and indicate

correctly the centre of a circle. He showed no evidence of apraxia, and dressing was correctly carried out, albeit slowly, as soon as adequate power returned to the left upper limb.

By eight weeks after admission he was behaving normally, and appeared to pay as much attention to the left as the right half of extra-personal space. His condition when he was discharged on 25.10.50 was satisfactory.

6. Mrs.E.D. Aet.69. Admitted 1.10.50.

History - For three weeks she had experienced difficulty in walking, and for one week she had suffered from dysarthria. Three days before admission she developed a left hemiparesis. She had no headache, loss of consciousness, ocular symptoms or fits.

Previous History - There was no history of neurological symptoms. For three years she had suffered from mild breathlessness on exertion, and ankle oedema had occasionally been noticed.

Family History - Her mother had died from a cerebral haemorrhage.

Social History - A housewife, she had led an active life to date.

Examination - A right-handed patient, she had a left hemiparesis. There was neither hemi-hypaesthesia nor hemianopia. Tactile inattention was

marked, and was present for light touch, pinprick, hot and cold objects, and vibration. There was no visual or auditory inattention, and no allachaesthesia. She was correctly oriented, and had neither autotopagnosia nor anosognosia. The cranial nerves were normal apart from the upper motor neurone left facial palsy.

Her pulse was regular, and the blood-pressure was 140/90 mm. mercury. The heart, however, was much enlarged, and evidence of previous hypertension was present in the form of a Grade 2 retinopathy. No signs of neoplasia were found in the chest or elsewhere. The blood Wasserman reaction was negative. Neither lumbar puncture nor X-rays of the skull were carried out, as it was felt that the patient had undoubtedly had a cerebral thrombosis involving the right internal capsule and thalamo-parietal projection. After making fair initial progress, she died quite suddenly on 23.10.50, and permission for autopsy was unfortunately firmly refused.

7. Mrs.A.A. Aet. 66. Admitted 9.10.50.

History - On 8.10.50 she developed left-sided weakness and collapsed, without injuring herself. She did not lose consciousness, and had no headache or convulsions, but her speech was impaired. She was admitted to hospital next day.

Previous History - None of relevance was obtained.

Family History - None relevant.

Social History - A housewife in quite comfortable circumstances.

Examination - She was an obese and somewhat apathetic patient. She was correctly oriented and quite clear as to her present and previous history. She stated that she was right-handed. She had a left hemiplegia and a left homonymous hemianopia. Hypaesthesia in all modalities was present in the left lower limb, and she was unaware of the left upper limb, denying its presence repeatedly despite its being pointed out to her. It seemed to have disappeared entirely from consciousness. Painful and other crude stimuli applied to it evoked no response. There was neither autotopagnosia for the left lower limb nor denial of its being paralysed. She had no allachaesthesia. It was impossible to test for the presence or absence of tactile inattention owing to the degree of hypaesthesia. The cranial nerves were normal apart from the upper motor neurone lesion affecting the left side of the face, and the hemianopia.

Her pulse was regular, and the blood-pressure was 220/112 mm. mercury. The heart was moderately enlarged, and the retinae showed Grade 2 hypertensive changes. The urine, consistently poorly concentrated, contained albumen and granular casts, but no leucocytes. The blood urea nitrogen was 14 mg./100 mil. The blood Wasserman reaction was negative. Lumbar puncture was not performed, and X-rays of the skull were not taken.

She lost her partial autotopagnosia and hemianopia within two weeks of admission, but some degree of hypaesthesia remained, though of lesser severity than that initially noted. Tactile and visual inattention appeared, and persisted till the day of her death on 1.4.51. Despite active physiotherapy, there had been no recovery of power.

Autopsy - This disclosed chronic pyelonephritis, left ventricular hypertrophy secondary to hypertension, and gross coronary and aortic atheroma. Recent subdural haemorrhage was present, especially on the right side. The brain on section showed a wedge-shaped area of infarction in the lower portion of the right corona radiata and upper portion of the right internal capsule, of some duration, as judged by encapsulation. The thalamus was normal. The thalamo-parietal connections, however, were obviously involved in the damaged area.

8. Mrs. J.W. Aet. 46. Admitted 23.10.50.

History - This patient gave a history of sudden onset of headache, vomiting and left-sided paralysis a few hours before admission. She did not lose her speech or suffer from convulsions.

Previous History - For some months she had experienced paroxysms of palpitations. She gave no history of rheumatic fever or of previous neurological symptoms.

Family History - None of relevance was obtained.

Social History - A housewife in moderate circumstances.

Examination - The patient was an obese woman, retching violently. She was afebrile. She had signs of mitral stenosis with apparent auricular fibrillation. There was no pulse deficit, and the blood-pressure was 100/85 mm. mercury. The heart was slightly enlarged (right ventricle), but there was no evidence of cardiac failure.

A right-handed patient, she had a left hemiparesis, with left hemi-hypaesthesia for all modalities, and a left homonymous hemianopia. She was correctly orientēd, and apparently not confused, but she paid little attention to the left side of the body, and announced that she had no paralysis. The hypaesthesia was not sufficiently severe to prevent testing for sensory inattention; and it was found that she had tactile inattention in all areas of the left half of the body. There was also hyper-reaction to painful stimuli in the same territory. The cranial nerves were normal apart from the left upper motor neurone facial palsy, and the hemianopia.

An electro-cardiogram revealed the presence of auricular flutter with varying block. No lumbar puncture was undertaken. It was considered that the patient had had a cerebral embolus affecting the right thalamus, internal capsule and thalamo-parietal projection

The anosognosia and hemianopia disappeared after a few days, but visual inattention succeeded the hemianopia. On 26.10.50 allachaesthesia was noted: it was present for some days. There was still no evidence of autotopagnosia, but the patient appeared to pay less attention to the left half of extra-personal space than the right. Spontaneous pain in the left arm and leg was moderately severe, and the patient announced that the affected limbs should be amputated.

During the subsequent ten days she had a number of delusions (e.g., she stated that she had spent the night travelling between Haddington and North Berwick) and also hallucinations of movement of the paralysed limbs. But she thereafter began to improve, and by 24.11.50 she was free from hypaesthesia, spontaneous pain and sensory inattention. Power had also returned to the affected limbs.

Convalescence was delayed by the occurrence of pulmonary emboli, but the patient was finally discharged, walking with the aid of a stick, on 16.2.51. Auriculectomy, or alternatively, valvotomy, were considered, but not ultimately advised.

The patient has been seen at intervals since discharge, and although still suffering from left-sided weakness, she has remained free from sensory symptoms and signs, or from any return of anosognosia.

9. Mrs. E.H. Aet. 60. Admitted 4.11.50.

History - Two days before admission, she temporarily lost consciousness, and on recovering her senses she found that weakness of the right side was present. She had no headache or vomiting. Transitory difficulty with speech was experienced.

Previous History - For three years she had suffered from breathlessness on exertion, and for two years she had noticed that her memory was failing.

Family History - There was none of relevance.

Social History - A housewife of adequate means.

Examination - The patient was right-handed. She had a right hemiparesis (the lower limb being little affected), right hemi-hypaesthesia, and a right homonymous hemianopia. There was impaired two-point discrimination and astereognosis in the right hand. She also had mild acalculia and finger agnosia, but no dysgraphia, despite weakness. Difficulty in distinguishing between right and left only occurred when the subject's or examiner's hands were crossed. Slight constructive apraxia was present. There were no abnormalities in the cranial nerves apart from the upper motor neurone right facial weakness and the hemianopia.

The pulse was regular, and the blood-pressure was 210/120 mm. mercury. The heart was moderately enlarged, and a generalised systolic murmur was heard. The retinae exhibited Grade 2 hypertensive changes.

No signs of congestive failure were present while the patient was resting. Examination of the other main clinical systems revealed no further points of note. The urine was normal. The blood Wasserman reaction was negative. A lumbar puncture was not performed, and no X-rays of the skull were taken.

This patient thus showed a partial Gerstmann syndrome, in addition to her other disabilities.

She improved rapidly spontaneously, and when discharged on 29.11.50 she had good recovery of power on the right side. There was no remaining evidence of hemianopia or hypaesthesia. She still showed hesitancy in calculation, and some difficulty in distinguishing fingers. Two-point discrimination and stereognosis were faulty in the right hand, although less so than when she was admitted.

It was considered that she had suffered from a thrombosis of the left middle cerebral artery, involving initially a wide area of motor and sensory cortex.

10. M.L. Aet. 50. Admitted 25.1.51.

History - On 19.1.51 he developed left-sided weakness, with associated headache and mental confusion, but he did not lose consciousness or have fits. He noted mild and transitory speech impairment.

Previous History - There was no history of neurological symptoms apart from headaches for some months.

He had also suffered from breathlessness on exertion for a similar period.

Family History - One of his brothers suffered from hypertension.

Social History - An artisan.

Examination - A right-handed patient, he had a left hemi-paresis, left hemi-hypaesthesia, and a left homonymous hemianopia. Well-marked tactile inattention was present for all modalities throughout the whole left half of the body. There was no evidence of either autotopagnosia or anosognosia. There were no other points of note in the nervous system. Speech was normal, and the patient was correctly oriented.

The pulse was regular, and the blood-pressure was 200/125 mm. mercury. The heart was much enlarged, the apex-beat being in the sixth interspace in the anterior axillary line. The sounds were normal, and there was no evidence of congestive heart failure. The retinae exhibited Grade 2 hypertensive changes. The urine was well concentrated, and contained no abnormalities. The blood urea nitrogen was 16 mg./100 mil. An electrocardiogram showed changes characteristic of left ventricular strain. An intravenous pyelogram was normal. The blood Wasserman reaction was negative, and X-rays of the skull revealed no abnormalities. No lumbar puncture was carried out.

It was considered that he had had a cerebral

thrombosis, secondary to hypertension, involving the right internal capsule and thalamo-parietal projection.

With rest followed by physiotherapy, this patient made a good recovery, although he was left with some residual weakness. Sensation returned to normal within three weeks of admission, apart from tactile inattention, which persisted for some weeks thereafter. The visual fields improved, but a small left lower quadrant homonymous defect was left. He did not, once vision began to improve, exhibit visual inattention. He was discharged on 9.4.51.

11. Mrs L.S. Aet. 62. Admitted 20.3.51.

History - It was learned from her doctor that three days before admission the patient developed confusion and dysphasia, without any sign of paralysis, loss of consciousness, vomiting or fits.

Previous History - She had suffered from a right hemiplegia some years previously (the exact date was unknown), and from a left hemiplegia in December 1950. From this she had made a good recovery.

In 1930 bilateral nephro-lithotomy was performed. For six years she had suffered from breathlessness on exertion, but there was no recent history of urinary symptoms.

Family History - None could be obtained.

Social History - A housewife in comfortable circum-

stances.

Examination - Initially she was much confused, dis-oriented for time and place, and almost speechless. She had residual signs of her former hemiplegias, without much muscular weakness. There was a sinus tachycardia (120/min.), the blood-pressure was 180/110 mm. mercury, and the apex-beat was in the sixth left inter-space, almost at the anterior axillary line. There was a triple rhythm (fourth sound). A Grade 3 hypertensive retinopathy was observed, and the urine was poorly concentrated, but sterile and free from pus. An electro-cardiogram showed changes indicative of myocardial ischaemia, but not of actual infarction. The blood urea nitrogen was 25 mg./100 mil., and the blood Wasserman reaction was negative.

Within two days of admission she became better oriented, and it was then possible to ascertain that she had a severe dysphasia, both expressive and receptive, dyslexia, dysgraphia, acalculia, finger agnosia, and difficulty in distinguishing between right and left. It was learned that she was right-handed. There was no evidence of hypaesthesia, hemianopia, sensory inattention or apraxia. She thus showed all the features of Gerstmann's syndrome, as well as motor dysphasia and evidence of former bilateral pyramidal lesions. It was considered that she suffered from advanced cerebral arteriosclerosis

secondary to hypertension.

Despite the fact that for a time her condition improved, she ultimately died on 30.4.51 as a result of a myocardial infarction which occurred on 20.4.51.

Autopsy - This revealed chronic pyelonephritis, advanced coronary arterial disease with recent anterior and posterior myocardial infarctions, and generalised severe cerebral arteriosclerosis. There were no macroscopic areas of cerebral haemorrhage or softening. The microscopic changes in all areas of the cerebral cortex were simply those of advanced cerebral arteriosclerosis - nerve-cell degeneration and secondary neuroglial overgrowth.

12. Mrs.M.L. Aet. 68. Admitted 2.5.51.

History - Six days before admission, she fell, striking the left side of the head. She did not hurt herself severely, and there was no loss of consciousness. On the day of admission she became confused and speechless, and could not use the right upper limb.

Previous History - None of relevance was obtained.

Family History - There was none of note.

Social History - She was a housewife in moderately comfortable circumstances.

Examination - Though conscious and apparently correctly oriented, she was aphasic and retarded. It was learned that she was right-handed. There was weakness

of the right side of the face and of the right upper limb, with increased tonus and deep reflexes. The right lower limb was not weak, and there was only slight exaggeration of tonus and deep reflexes, but the plantar response was extensor. The pupils and visual fields were normal, as were the optic discs. Apart from the upper motor neurone right facial palsy, the cranial nerves were normal. As regards sensation, the patient seemed to feel painful stimuli on the right side of the body as intensely as on the left. More detailed sensory testing was at this juncture impracticable.

There was no cardiac arrhythmia. The blood-pressure was 230/135 mm. mercury, and the heart was moderately enlarged, although there was no evidence of cardiac failure. A Grade 1 hypertensive retinopathy was present.

Lumbar puncture yielded slightly blood-stained fluid under normal pressure. Apart from some increase in the protein content, it was biochemically normal, and serologically negative. The blood Wasserman reaction was also negative. X-rays of the skull revealed a linear fracture in the left parieto-occipital region.

The patient's condition improved spontaneously, with rapid return of power to the face and right upper limb. Paresis was, however, succeeded by apraxia, and aphasia by a mumbling dysphasia. Evidence of agraphia, acalculia and dyslexia appeared, although there was neither finger agnosia nor

inability to discriminate between right and left. No abnormalities of sensation were discovered.

When discharged on 9.6.51, she still had a gross receptive dysphasia, but she was able to recognise her own property, and to read and obey instructions. The apraxia had disappeared, and she could correctly perform simple calculations. At no time was there evidence of impaired appreciation of extra-personal space or loss of topographical memory.

The nature and extent of the lesion were debatable. The possibility of a subdural haematoma arose initially, but the subsequent natural history of the case rendered such a diagnosis unlikely. It was felt that the patient probably suffered from a rather widespread thrombosis - secondary to trauma - affecting most of the cortical branches of the left middle cerebral artery. She had certain components of a Gerstmann syndrome as well as apraxia and receptive dysphasia.

13. Miss J.F. Aet. 56. Admitted 25.6.51.

History - Following a period of several weeks of tiredness, giddiness and headaches, she developed left-sided weakness late on 24.6.51. There was no loss of consciousness, and she had no convulsions. Speech was little impaired.

Previous History - There was none of relevance.

Family History - There were no points of note.

Social History - A factory worker.

Examination - An obese, right-handed woman, she had a left hemiplegia, the face, palate and tongue being involved. Although there was no paralysis of ocular muscles, she looked mainly towards the right, and had a left homonymous hemianopia. She also had a left hemi-hypaesthesia, with some spontaneous pain in the limbs on the left side. Although correctly oriented and otherwise rational, she at first had a definite autotopagnosia for the left half of the body, amounting to a disappearance of this territory from consciousness. There was no evidence of anosognosia, and no tactile inattention or allach-aesthesia.

General clinical examination suggested the presence of arteriosclerosis, but there were no other points of note. The blood-pressure was 150/90, and the cardiac rhythm was regular. The blood Wasserman reaction was negative, and the results of lumbar puncture and skull X-rays were normal.

She made slow but definite progress. Autotopagnosia disappeared within two days of admission, but power did not return to the left upper limb until 29.7.51, although spontaneous movements of the left lower limb were noted by the end of June. The extent of the hemianopia diminished, but a permanent partial field defect was left. Hypaesthesia and spontaneous pain were present until 10.8.51. Sensory inattention was never noted. When she was discharged some weeks later, she had residual hemi-

plegic signs and the field defect already mentioned.

It was considered that she had had a cerebral thrombosis involving the right internal capsule and the thalamo-parietal projection. The occurrence of spontaneous pain rendered involvement of the right thalamus likely, in addition.

Discussion.

It is appreciated that this study would have been more complete had the clinical course of the majority of patients been different. Of the thirteen patients studied, five died, but in only two of these cases was autopsy permitted (Cases 7 and 11). In one of the others (Case 2), however, an ante-mortem diagnosis of neoplasm involving the left parietal lobe was made, and confirmed at craniotomy. As regards exact localisation of the lesions in most of the cases, therefore, little can be said; but reports already published in the literature on parietal lobe syndromes allow a reasonable surmise to be made concerning the probable distribution of lesions in the patients who survived.

Age

The ages of the thirteen patients varied from forty-six to sixty-nine years, the average age being 60.8 years. Eight patients were aged sixty years or over.

Aetiology

The aetiological cause of the lesions in the patients studied is shown in Table I. The considerable preponderance of hypertensive arterial disease as a cause will be noted. In this series, hypertension was diagnosed in patients who had a diastolic pressure of 100 mm. mercury or more, or who, although having a lower diastolic pressure, showed undoubted hypertensive retinal changes; the presumption in the

latter patients being that the blood-pressure had previously been higher, and had fallen as a result of cardiac failure.

Table I:

(The figures in the columns indicate the serial number of the cases).

	Hyper- tension.	Athero- sclerosis.	Tumour.	Embolus.	Syphilis.
Case 1.		3.	2.	8.	Nil.
4.		5.			
6.		13.			
7.					
9.					
10.					
11.					
12.					
—		—	—	—	—
8.		3.	1.	1.	Nil.
—		—	—	—	—

The series is too small to permit any comparison between different aetiological factors, but tumour probably figures relatively too seldom, since cases suggestive of cerebral neoplasm might tend to be sent direct to a Neuro-surgical Unit. Similarly, experience acquired in London since the above cases were studied has suggested that there neuro-syphilis is relatively, as well as absolutely, much more common than in Edinburgh.

Sex.

Nine patients were female, four were male. The series is too small to attach any significance to such a finding.

Syndromes.

The frequency of different manifestations of parietal disorders is shown in Table II.

Table II.

Sensory Inattention.	Gerstmann Syndrome.	Anoso- gnosia.	Autotop- agnosia.	Visual- spatial agnosia.
Case 3.	2.@	1.@	1.@	3.
5.	4.	7.@	13.	4.
6.@	9.	8.		5.
7.@	10.@			8.
8.	12.			
10.				

@ - Died.

Sensory Inattention.

Six cases (Cases 3,5,6,7,8 and 10) showed tactile inattention, and four of these (Cases 3,5,7 and 8) had also visual inattention. In none of the patients was auditory inattention noted. Critchley (1949) has reviewed very fully the phenomenon of tactile inattention with special reference to parietal lesions, and has pointed out that such inattention may be demonstrated quite often if specially sought. This contention receives some support from the present series (six cases out of thirteen with "parietal" signs, and out of a total of fifty-five patients having either cerebral vascular accidents or cerebral tumours). Of the patients having tactile inattention, four (Cases 5,7,8 and 10) had hemihypaesthesia as well, while two (Cases 3 and 6) had no detectable hypaesthesia, despite Critchley's

(loc. cit.) contention that it is doubtful if tactile inattention ever occurs in the absence of some depression of sensation. Critchley has also stated "It is probably safe to suggest that if all patients with imperception for one body-half were to be subjected to appropriate testing, a tactile inattention would also be found." With this proposition I am unable to agree, as neither of the two cases of autotopagnosia in this series showed tactile inattention, despite repeated careful testing.

Of the four cases of visual inattention, two (Cases 7 and 8) had hemianopia preceding the inattention.

As regards the localising value of sensory inattention, the majority of reported cases (e.g., Pinéas (1931), Bender and Furlow (1945), Nathan (1946), Ross and Fountain (1948)) have occurred in association with parietal lesions. Nevertheless, cases do from time to time appear such as those of Bender (1945) wherein sensory inattention is present as a result of spinal lesions. Alternatively it may be found in association with cerebral lesions outside the parietal lobes, in a setting of intellectual confusion.

Concerning the nature of sensory inattention, Critchley's (loc.cit.) explanation seems the most convincing in the present state of knowledge. Writing of tactile inattention, he has rightly pointed out that the phenomenon cannot be explained along

purely physiological lines, on account of its variability from moment to moment, and the possibility of extinguishing it by repeated testing or the use of very strong stimuli. He has suggested that tactile inattention may be explained by regarding the problem as one of rivalry between two contesting stimuli, transcending what occurs in the normal experimental subject (in whom "of simultaneous pains in two places the lesser is obliterated by the greater" as Hippocrates wrote). "A stimulus applied to a bodily segment of high perceptual threshold fails to attain conscious level as it is distracted by a rival stimulus synchronously applied to a segment with a lower perceptual limen" (This assumes that all cases of tactile inattention have also hypaesthesia, however).

Other writers (e.g. Bender (1945), Reider (1946)) have argued that inattention cannot explain the phenomenon, because it may continue even after the patient has been instructed to focus his attention on the affected limb. As Critchley points out, this argument is fallacious as attention in its fullest sense requires the active ideational (as well as sensorial) excitation of the appropriate cortical centre, and such attention may not be possible of attainment in patients with parietal lesions.

Critchley concludes "In the genesis of tactile inattention, however, disease of the parietal lobes plays a significant and highly specific role. This

may be explained along three lines. (1) The role of the parietal cortex as an organ of local attention (Head and Holmes); (2) the factor of impairment of sensibility leading to the rivalry of contending double percepts of unequal strength; and (3) the importance of the parietal lobe as the storehouse of past impressions and hence in the building up of a body scheme or image de soi. A severe parietal lesion will therefore be followed by defective attentiveness, sensory impairment, and a tendency more or less to neglect the affected side of the body - three considerations which are important in setting the stage for the appearance of tactile inattention." The same general considerations would appear to apply to the problems of visual and auditory inattention, the difference being in the areas of cortex or subcortical connections affected.

Gerstmann's Syndrome.

Gerstmann (1942) says that his syndrome of finger agnosia, agraphia, acalculia, and inability to distinguish between right and left (1924, 1927, 1940), "usually caused by a local process affecting the lower part of the parietal lobe, or specifically the angular gyrus in its transition to the second occipital convolution, seems to be the only disturbance of the body scheme proper which can be found

as a result of a focal cerebral lesion." He refers to the dominant parietal lobe.

He says that reports on his syndrome are numerous, whereas autotopagnosia is rare. Accounts of cases of his syndrome are certainly numerous if one allows partial examples - e.g., finger agnosia and agraphia alone. Neilsen (1938b), in an exhaustive discussion of the syndrome, pointed out that Lange (1933) had drawn attention to a case of Conrad's in which finger aphasia occurred, but no agraphia, and to a case of Schlesinger's with finger apraxia as opposed to agnosia; other cases showed finger agnosia alone, without the other specified defects of the original syndrome. Engerth (1933) reported that at times finger agnosia is associated with severe defects of drawing, especially of the face and hands. He considered the latter defect an indication of autotopagnosia for these parts.

Schilder (1931) drew attention to the fact that up to that time, five disturbances of finger function had been reported in cases of "Gerstmann's syndrome". These he thought had a localizing value. These disturbances were as follows -

- (1) Optic finger agnosia, due, he thought to lesions nearest the occipital pole.
- (2) Finger agnosia, resulting from lesions in the transition zone between the angular gyrus and the second occipital convolution.
- (3) Constructive finger apraxia, resulting from lesions between the region for finger agnosia and the

supramarginal gyrus.

(4) Apraxic disturbance in finger selection, due to lesions in the supramarginal gyrus.

(5) Finger aphasia, from lesions of the extension of Wernicke's zone.

Unfortunately, this scheme is altogether too tidy. And although it is true that often no proper attempt is made to decide whether a patient has really finger agnosia, or one of the four other conditions listed by Schilder, such precise localisation on clinical grounds does not always fit with the pathological findings.

In the present series, five patients showed the syndrome wholly or in part (Cases 2, 4, 9, 10 and 12). Two of these died (Cases 2 and 11). In Case 2, no autopsy was performed, but at craniotomy a large glioblastoma multiforme involving the left parietal lobe was demonstrated. In Case 11, autopsy disclosed only generalised cerebral arteriosclerosis, despite careful search for a focal parietal lesion. The latter case, like some others reported, helps to dispose of the concept of inevitable exact localisation, although it appears to be true that many examples of the syndrome have displayed lesions in the area suggested by Gerstmann.

This syndrome is found only with lesions of the dominant hemisphere. The suggestion has been made that autotopagnosia is the equivalent disturbance of function resulting from a lesion in the non-dominant hemisphere. In so far as the differing

functions of the two hemispheres relating to the body image are concerned, there is a certain amount to be said in favour of this concept; but for those who wish to hold firm to the notions of exact cerebral localisation, it may be recalled that autotopagnosia frequently arises as the result of lesions more inferiorly placed, i.e., nearer the thalamus. An interesting link between major and minor hemispheres is, however, provided by three cases on record. First is the case of Hermann and Pötzl (1926). Their patient, who was ambidextrous, had a right-sided lesion, and in addition to displaying a typical Gerstmann syndrome, he had autotopagnosia for his left limbs. As Neilsen (1938b), who supplies the second case, remarks, it is interesting to speculate what a left-sided lesion would have produced in this case. Neilsen's patient was a woman suffering from hypertension, who, as a result of a cerebral vascular accident developed a right hemiplegia, right homonymous hemianopia, and right hemi-hypaesthesia; in addition, she had allocheria, allachaesthesia, anosognosia for the paralysis of the lower limb but not the upper, a feeling that the right upper limb was "wrong" (possibly autotopagnosia), and a Gerstmann syndrome. There was no history of left-handedness, yet undoubtedly a combination of major and minor hemisphere signs developed.

The third case is that reported by Sandifer (1946),

also quoted below in the section on anosognosia, in which a patient, stated to be right-handed, with a right hemiplegia, had a partial Gerstmann syndrome (which one might expect with a lesion of the dominant hemisphere), and anosognosia.

Neilsen agrees with others that a reason for the differing symptomatology of major and minor hemisphere lesions is that the major parieto-occipital area is one of correlation, in which the hand is converted from a discrete organ to a tool for the service of the whole body. With training, resulting from "handedness", the cruder elements of body-image organisation disappear from the activity of the dominant hemisphere. Neilsen continues " in the same region is a laterality co-ordinating area, a lesion of which causes loss of the sense of right and left, and even entire loss of the sense of direction. The more closely the lesion approaches the occipital pole, the more of a visual element one finds in the disturbance. The nearer the lesion approaches the parietal region, the more of a sensory element there appears. The more closely the lesion approaches Wernicke's zone, the more of a language, or symbolic element one finds." With the burden of his contentions there will be general agreement, always allowing that exact localisation tends to be a practice of fitting facts to theories rather than the converse.

Anosognosia.

Three patients showed anosognosia for hemiplegia (Cases 1, 7 and 8). Case 7 denied paralysis of the upper limb only, and realised that the lower limb was paralysed. Such a dissociation is occasionally met. Neilsen (1938b) describes a patient who was aware that her upper limb was weak, but who did not realise that her lower limb was paralysed.

Two of these patients died, but only one came to autopsy (Case 7). This disclosed a wedge-shaped infarct of the right lower corona radiata and upper internal capsule, but no lesion of either the thalamus or parietal cortex, though a subdural haemorrhage of apparently recent onset overlay the parietal cortex.

Two points arise for consideration, namely,

- (1) What is the nature of anosognosia?
- (2) Has it a localising value?

These matters are so closely inter-related that if they are discussed together needless repetition will be avoided.

The term anosognosia has by popular usage developed a limited connotation of imperception of hemiplegia, although in fact the earliest quoted example of anosognosia in its wider sense was a case of unawareness of (cortical) blindness (von Monakow, 1885) (later confirmed by Déjerine and Vialet, 1893). Unawareness of left hemiplegia was described by Pick (1898), but Anton (1898, 1899) was, to quote

Gerstmann (1940) "the first to emphasise the problem of non-perception of disease with certain organic defects, and to show its connection with localised cerebral lesions, describing the condition in cases of left hemiplegia as well as in those of cortical blindness or deafness." His conviction of the localising value of anosognosia (for hemiplegia) was upheld by Babinski (1914). Redlich and Bonvicini (1907,1908,1911), however, dissented from these views, and believed that anosognosia developed as a result of a Korsakow-like state.

Barkman (1925), reporting a case of left hemiplegia with anosognosia, noted a lesion of the right thalamo-parietal projection and right thalamus. He expressed the opinion that anosognosia resulted from failure of sensory impulses to reach consciousness. Neilsen (1938b) agreed with this conclusion.

Pötzl (1925), described two cases of anosognosia for left hemiplegia and delusions related to the left half of the body. One patient had had a fresh haemorrhage adjacent to a cystic area in the right parietal lobe, with softening of the right thalamus, while autopsy on the other case revealed a softening in the right parietal lobe and another in the right thalamus. Pötzl considered that co-incidental lesions in the thalamus and the minor parietal lobe were responsible for anosognosia. He evidently overlooked a report by Müller (1905), who described a case of anosognosia for left hemiplegia wherein at autopsy softening of the right supramarginal

gyrus, the anterior part of the right internal capsule, and the corpus striatum, were found, but no thalamic lesion.

Pinéas (1926), reporting five cases of anosognosia for left hemiplegia, described two cases wherein no thalamic lesion was discovered. In one, softening in the right supramarginal and angular gyri, as well as in the insula, caudate nucleus and putamen, was noted; in the other, there was softening in the area of the right central convolution, the parietal lobe and thalamus both being intact.

Anosognosia for hemiplegia has received further attention, notably from Von Hagen, Ives and Neilsen. Von Hagen and Ives (1937) reported six cases, with one autopsy. The post-mortem examination disclosed an abscess involving the right thalamus and thalamoparietal projection. The same authors (1939) described two further cases, both fatal. One case, in which left hemiplegia, sensory impairment and left homonymous hemianopia were present, was found at autopsy to have haemorrhages in the right corpus striatum just lateral to the thalamus. The other, with in life left hemiplegia and impaired sensation in the left half of the body, at autopsy had softening of the right parietal lobe (supramarginal and angular gyri and inferior portion of the superior parietal lobule) extending down to the corona radiata but not to the thalamus.

Ives and Neilsen (1937) reported two cases of anosognosia for left hemiplegia with delusions con-

cerning the left limbs. At autopsy, one patient had vascular lesions in the right thalamus, the retrolenticular part of the internal capsule, and the thalamo-parietal projection. The other patient had extensive softening of the right parietal lobe, affecting especially the supramarginal and angular gyri.

Sandifer (1946), writing of three cases of imperception of disease, described two patients having anosognosia for hemiplegia. The first, a right-handed female aged sixty-six years, had left hemiplegia, a left homonymous hemianopia, left autotopagnosia, and possibly agnosia for the left half of extra-personal space. Testing for sensation in the left half of the body produced appropriate emotional responses, but a denial of pain having been felt. Autopsy showed softening of an area of the right cortex, bounded by the precentral gyrus anteriorly, the angular gyrus posteriorly, and the superior temporal sulcus below. Section revealed softening extending from the thalamus to the inferior frontal gyrus and the superior temporal gyrus. The other patient, a right-handed female aged sixty-nine years, was of special interest, in that two years after a transient left hemiplegia, she developed a right hemiplegia with apparent anosognosia. She had some difficulty in distinguishing between right and left, and when asked to use her right hand, employed her left one instead. Gross acalculia and object agnosia

were present, but no finger agnosia. There was no hemianopia, autotopagnosia, or agnosia for the right half of extra-personal space. This case provides an interesting link with Gerstmann's syndrome.

Sandifer's third case was one of agnosia for complete blindness, with confabulation and complete disorientation, the result of a recurrence of an oligodendroglioma in the left temporal lobe, the primary growth in the left occipital lobe having been removed previously. The optic nerves were grossly distracted.

The author, discussing these three cases, believed that anosognosia arose from a combination of an organic lesion and a confusional state. The former, he considered, might exist at any level of the nervous system, provided there were an appropriate intellectual defect accompanying it. He goes on to suggest that possibly the ability to re-experience past events depends on the capacity for forming and utilising engrammatic records of current experience. Brain damage preventing the formation and utilisation of such records, as from destruction of association fibres, will prevent the retention in memory of some forms of current experience, and the evocation of some varieties of past experience. Stengel (1944) has likewise attached great importance to dementia and clouding of consciousness in the genesis of anosognosia. While no doubt such explanations may be acceptable in some cases of anosognosia, it is the experience both of other authors and myself

that the majority of cases of anosognosia develop in the absence of a confusional state. Recently Weinstein and Kahn (1950), as a result of a study of twenty-two tumours in all parts of the brain, have suggested that anosognosia has no focal diagnostic value, but is "a manifestation of the patient's desire to be well." Without going to so dogmatic a limit, a similar theory was advanced by Schilder (1935) as one explanation of anosognosia, but his reasoning was criticised by Gerstmann (1942) on the grounds that it is odd that the instinctive urge should operate only in the case of lesions of the minor hemisphere, and not those of the major.

Roth (1949), describing two cases of body-image disorder due to lesions of the right parietal lobe, discusses the neurological lesions in anosognosia for hemiplegia and goes so far as to say "Such a lesion" (of the right parietal lobe) "has been a constant finding in all cases of anosognosia for left hemiplegia examined post-mortem, even when the syndrome has been manifested in a setting of mental confusion." A study of Table III suggests that this contention is justified, as long as it is admitted that in many cases the responsible lesion is situated subcortically, rather than in the parietal cortex.

Table III:- Sites of Lesions in Cases of Anosognosia

(The list is not claimed to be exhaustive).

<u>Author</u>		<u>Site of Lesion</u>		
		Thalamus	Thalamo-parietal projection	Parietal Cortex
Anton	(a)	No	Yes	Yes
	(b)	No	Yes	Yes
Müller		No	Yes	Yes
Pötzl	(a)	Yes	No	Yes
	(b)	Yes	No	Yes
Barkman		Yes	Yes	No
Pinéas	(a)	No	Yes	Yes
	(b)	No	Yes	No
Von Hagen and Ives	(a)	Yes	Yes	No
	(b)	No	Yes	No
	(c)	No	Yes	Yes
Ives and Neilsen	(a)	Yes	Yes	No
	(b)	No	No	Yes
Sandifer		Yes	Yes	Yes
Roth		No	Yes	Yes
Guild		No	Yes	No

It appears, therefore, that we must be careful to distinguish anosognosia due to clouding of consciousness or dementia, an aetiology proposed notably by Redlich and Bonvicini (1907, 1908, 1911), Stengel (1944) and Sandifer (1946), from the state occurring in patients with no such mental defect. Both types must be distinguished in turn from anosodiaphoria, or lack of concern over the presence of disease, a symptom which may arise as a result of lesions of the subordinate parietal lobe. The former variety of anosognosia may apparently be associated with lesions in differing areas; a study of reported cases suggests that the latter variety may arise from either lesions of the parietal cortex of the subordinate hemisphere, or from damage to the thalamo-parietal projection on the same side - or both, but probably not from isolated thalamic damage.

Gerstmann (1942), writing about right-handed individuals, comments that while it cannot be denied that divergences in the relative changes observed at autopsy still exist, there is, on the other hand, increasing evidence that anosognosia is most likely to result from lesions affecting the right thalamus, the right thalamo-parietal projection, or the parietal cortex of the minor hemisphere. Moreover, the fact (he thinks) seems to emerge that lesions nearer to or within the structure last-named, particularly those in the cortex about or below the right interparietal fissure, are more apt to produce the psycho-

logically complex manifestations of the condition than those further away from it, nearer the thalamus "which may, or may not, be immediately involved."

Despite Gerstmann's statement, as previously indicated it seems doubtful whether lesions of the thalamus alone can lead to anosognosia.

Roth (loc.cit.) has aptly said that no complete explanation of anosognosia is yet possible "in our present state of ignorance about the mode of evolution and physiology of the body image" Attempts to explain the condition on a basis of sensory loss have been made (Barré (1923): Barkman (1925)), but as Roth properly remarks "the patient usually retains adequate sensory and intellectual means for discovering his hemiplegia." Again, hemianaesthesia or hemihypaesthesia in conjunction with hemiplegia is not uncommon, while anosognosia for hemiplegia is much more rarely encountered, and sensory loss in cases of anosognosia may be slight or absent. Roth rightly says "When a patient ignores part of his body, his condition is no longer accountable in terms of sensory loss alone. For such behaviour is delusional, whether or not he expresses delusional ideas, and must be based on a defect of an agnostic kind."

Meige (1914), quoted by Neilsen (1938b) believed that anosognosia was conditioned by autotopagnosia (a contention which numerous cases of anosognosia without autotopagnosia, including two of the present series, refute). Neilsen points out that if anosog-

nosia and autotopagnosia are considered to result from delusional states, it should be realised that the delusions are of different orders - anosognosia on such a theory representing a desire to escape from the reality of a paralysis, while autotopagnosia fulfils no such wish, but is merely inconvenient.

Roth (loc. cit.) may again be quoted, with approval "However, it becomes especially apparent where there is little or no generalized intellectual disturbance, that it is difficult to account for the syndrome" (anosognosia) "without postulating an additional and possibly fundamental factor of agnosia for the body, in consequence of which the patient's orientation with respect to his body, is disturbed in a specific way, and he is no longer able to relate himself to it correctly."

"Even confused and demented people do not always deny a painless hemiplegia, and it would seem reasonable to assume that a specific, focal disorder of cognition, arising from a disorganization of the body scheme, makes its contribution to the genesis of the disorder in all cases."

This seems to be an eminently reasonable conclusion, in the light of our present knowledge.

Autotopagnosia.

Two patients displayed autotopagnosia (Cases 1 and 13). In each case, the condition seemed to consist of a disappearance of the half-body from consciousness, as the defect was not abolished by calling attention to the presence of the limbs on the affected side. Case 1 died, but unfortunately no autopsy was allowed.

As with anosognosia, two points principally arise,

- (1) What is the nature of autotopagnosia?
- (2) Has it a localising value?

Autotopagnosia is rarer than anosognosia, and case-reports with accounts of post-mortem examinations are relatively few. Neilsen (1938a) reported five cases with autotopagnosia or allied disorders:

- (1) A patient who tended to forget the left side of the body. A large glioblastoma multiforme in the right parietal region, with involvement of the thalamo-parietal projection, was found at autopsy.

- (2) A patient with amnesia for the left limbs and left homonymous hemianopia. At autopsy, softening in the right parietal region, with destruction of the supramarginal gyrus and involvement of the angular gyrus, was discovered.

- (3) A patient with left autotopagnosia. Post-mortem examination disclosed a haemorrhage in the right external capsule extending from the anterior border of the insula to the pulvinar.

- (4) A patient who denied the presence of the left limbs. Extensive softening in the territory

supplied by the right middle cerebral artery, but sparing the thalamus, was demonstrated at autopsy.

(5) A patient with delusions concerning a paralysed left arm. Post-mortem examination revealed thrombosis of the terminal branches of the right middle cerebral artery, with softening of the right thalamo-parietal projection.

The same author, discussing the nature of autotopagnosia in a further paper (1938b), has suggested that three degrees of psychic disturbance may be recognised:-

(1) Episodic amnesia for the half-body. This constitutes a disappearance of the limbs from attention rather than from consciousness, since the defect is abolished by calling the patient's attention to the limbs.

(2) Disappearance of the half-body from consciousness. This "negative" state is to be distinguished from

(3) the "positive" state of feeling of absence of the half-body. This may be a sensation only, being rejected by the patient on the basis of his visual sensations (von Pap (1934), Koch and von Stockert (1935)), or an actual delusion of absence (Zingerle (1913)).

Referring to its localising value, Neilsen (1938b) considered that the essential lesion responsible for autotopagnosia of any variety was situated in or above (i.e., in the parietal cortex) the thalamo-parietal projection on the minor side.

Evidence in support of this view may be obtained from a study of the cases described in his earlier paper (1938a), as summarised above. His distinction between autotopagnosia as a disorder of the psyche, and anosognosia, as resulting from a failure of sensory impulses to reach consciousness, however, appears to be unsound.

Schilder (1935) has analysed autotopagnosia from a different point of view:

- (1) Autotopagnosia due to defective sensory input or appreciation.
- (2) Autotopagnosia due to motor impulses to turn the body model, and with it the sensations, to the opposite side.
- (3) Autotopagnosia due to a motor tendency to look away (i.e., to the opposite side).
- (4) Autotopagnosia due to a tendency to neglect sensations in one half of the body.

It is difficult to believe that, however defective sensory input from one half of the body is, a patient not demented or confused could not still be aware of his body as a whole; enough sensation from the sound side and through the eyes would remain.

A failure of sensory appreciation at an apperceptive level is a more convincing explanation, as is Schilder's fourth cause, an inner tendency to neglect sensations from one half of the body. The latter might be interpreted as an organic repression in cases where a co-existent hemiplegia was distasteful,

and in such cases one might sometimes expect to find also anosognosia for hemiplegia. It is perhaps not difficult to understand how in confused patients a homonymous hemianopia might aid the formation of a delusional system rejecting the existence of the ipselateral half-body. Schilder rightly points out that right-handed people tend habitually to pay less attention to the left half of the body than the right, especially as far as the upper limb is concerned, and such "relative neglect" may be intensified by a focal lesion.

One might expect that patients whose autotopagnosia appeared to be dependant on a tendency to neglect one half of the body would simultaneously pay little attention to the equivalent half of extra-personal space. Yet as Gerstmann (1942) has said, such a combination is very rare. McFie et al. (1950) described six cases of unilateral spatial agnosia, in none of which was autotopagnosia present. Neither of the cases in the present series showed such a combination, and conversely, the four cases having visual-spatial agnostic disorders did not suffer from autotopagnosia.

Of the two cases of autotopagnosia reported here, Case 1 had both considerable hypaesthesia and disorientation. The autotopagnosia was probably due to both impaired sensory input and appreciation, and confusion. Case 13 had hemi-hypaesthesia and a homonymous hemianopia, but was well oriented and not



confused. The symptom in this case was not abolished by pointing out the "disavowed" limbs, and therefore represented a disappearance of the left side of the body from consciousness. Although hypaesthesia and hemianopia were no doubt contributory factors, they did not explain the entire symptom, as the affected limbs could be felt by the sound ones, and seen when brought into the intact half field of vision.

On the basis of the rather scanty case-material hitherto available, it appears that in patients who are not confused, autotopagnosia, whether this term refers to amnesia for half of the body, or the disappearance of the half-body from consciousness, may normally point to a lesion of the thalamo-parietal projection or the parietal cortex (or both) of the subordinate hemisphere. Similar lesions may produce anosognosia, and it is not clear in the light of our present knowledge, why sometimes one, sometimes the other symptom, but rarely both simultaneously, should so arise. There is so far insufficient evidence as to whether the condition has any localising value in those patients who have a feeling of absence of half of the body.

Disorders of Space Perception and Allied Disorders.

Before discussing the patients who exhibited these disorders, it is worth emphasising that such syndromes have recently begun to receive increasing attention. Of diverse nature, and occurring singly or in combination, they are still imperfectly understood. Perhaps the most important initial study was that of Marie et al. (1922), who described a condition closely related to what has come to be known as visual-spatial agnosia, under the term "planotopokinesia" - a syndrome referred to by Lange (1936) under the clumsy title of "apractognosia for spatial articulation". Lhermitte (1933) wrote of "a circumscribed dissolution of la pensée spatiale". Important contributions to our knowledge have recently been made by Brain (1941, 1945), Paterson and Zangwill (1944), Neilsen (1946), Hécaen and Ajuriaguerra (1949) and McFie et al. (1950). The last-named authors, discussing eight cases of visual-spatial agnosia, analysed these disorders in detail. The more important symptoms they noted were:-

- (1) Unilateral spatial agnosia - A tendency to neglect (the left) half of visual space. In none of the six of their patients who showed this defect was autotopagnosia present, as already noted.
- (2) Apraxia for dressing. Difficulties in dressing appeared to be due to confusion between top and bottom, back and front, and right and left, with reference to the garments. The authors point out

that the "apraxia" is really conditioned by a form of spatial agnosia.

(3) Visual-constructive disabilities - Disorders in the appreciation of higher spatial relationships and the performance of simple constructional tasks under visual control - e.g., in drawing, copying patterns with paper and scissors, or with matches in two and three dimensions, the use of Koh's blocks. Here again failure in performance depended on a form of spatial agnosia.

(4) Loss of topographical orientation. This disorder, generally episodic, was not, in one case, related to agnosia for (the left) half of space. It must be distinguished from

(5) Loss of topographical memory - an inability to describe correctly familiar routes and buildings, despite the absence of aphasia.

To some extent these defects may be inter-related, as for example a patient who may have both loss of topographical orientation or topographical memory, and an inability in the visual-constructive sphere correctly to reproduce plans of well-known houses or streets, or maps. Patients tending to neglect one half of extra-personal space may equally in the visual-constructive sphere pay no attention to the equivalent half of a drawing, map or model; or in the topographical sphere, they may consistently turn always towards the retained "half of space", with at times a consequent form of disorientation. The

important link is always the visual-spatial agnosia. This agnosia may frequently be associated with homonymous hemianopia, but is not invariably so. The association was noted in six cases out of eight reported by McFie et al.

Four patients in the present series suffered from forms of visual-spatial agnosia (Cases 3,4,5 and 8). Case 8 demonstrated merely a lack of attention to extra-personal space for about ten days. This was combined with a similar lack of attention to the ipsilateral (left) half of the body, but no real autotopagnosia. She also demonstrated anosognosia for her left hemiplegia, and had left homonymous hemianopia, left hemi-hypaesthesia and sensory inattention. It seems likely that her inattention to the left half of extra-personal space (and to the left half of the body) were the result of the hemianopia, with some clouding of consciousness. The disorders of spatial appreciation disappeared when full vision was regained, but as it happened, the clouding of consciousness also passed off at this time. Sensory inattention persisted considerably longer.

Cases 3 and 5 demonstrated more persistent disorders of interest in that they occurred in the absence of any other defects apart from sensory inattention. Neither patient suffered from autotopagnosia. The infrequency of such an association is one of the most interesting, as well as the most puzzling features

of such "parietal" conditions. Case 5 is of interest in that, although left-handed, he showed a disorder of left extra-personal space appreciation as a result of a lesion on the dominant side. It is true that his disability was comparatively slight, but it was quite definite. Brain (1941), discussing right-handed patients showing loss of topographical orientation, states " No corresponding disturbance has been reported as a result of a lesion of the left hemisphere" (i.e., the dominant one), but the same author (1945) has said that spatial disorientation and kindred disorders are to be ascribed to a lesion in the dominant hemisphere. Such a view has also been expressed by other writers (Neilsen (1946), Duke-Elder (1949), Hécaen and Ajuriaguerra (1949)). McFie et al. (loc.cit.), however, point out that the majority of cases wherein massive loss of topographical orientation has been reported showed either bilateral lesions, or a lesion of the minor hemisphere. It must be admitted that Case 5 had been made to write with his right hand, and therefore was to some extent ambidextrous. One would in his case have expected to find a reversed Gerstmann syndrome with marked dysgraphia, since the transfer of calligraphic dominance can never be a natural activity, yet such was not the case. This patient showed no evidence of loss of topographical memory, but such a disability, as revealed by inability to describe familiar routes or to draw (or

copy) plans and diagrams, was demonstrated by Cases 3 and 4, the latter patient having also a Gerstmann syndrome. Both these patients were right-handed. Both were asked, at intervals, to draw plans of their homes, and to copy maps of Scotland. Execution was initially exceedingly faulty, particularly in the drawings from memory, but it slowly improved, though neither patient at the time of discharge from hospital, or after several months' follow-up, could produce accurate plans, copies of maps, or drawings of such common objects as daisies. Each patient displayed a consistent neglect of the left half of their drawings. Daisies would be rendered thus -



Case 3, in copying a free-hand, simplified map of Scotland, would often leave the west coast quite incomplete. When asked to indicate the positions of the principal cities, he frequently placed them in the sea to the east (or right) of the land.

Both of these cases recovered, so that no absolute statements on localisation of lesions can be made. Certain tentative conclusions can, however, be reached. Case 3 was right-handed and had a left hemiparesis, without organic evidence of any left cerebral lesion as well. The presumption is that

his disorder of spatial perception resulted from a lesion of the right (minor) hemisphere. Case 4 was also right-handed, and had a Gerstmann syndrome. It is possible, of course, that she had more than one cerebral lesion, but if only one was present, her topographical disorder resulted from a lesion of the left (major) hemisphere.

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"Parietal" Lesions in General.

The development of our present attitude towards "parietal" lesions may be briefly sketched. The first step was the discovery of certain main symptoms such as anosognosia or autotopagnosia. Cases were gradually gathered together, and with the growth of case material, and particularly of the findings of autopsies, a fresh stage was entered - that of trying to relate symptoms or syndromes to exactly localised lesions. Experience, somewhat slowly accumulated during the last thirty years, has led us in turn to our present attitude, which is that we must acknowledge that not infrequently clinical symptoms and signs cannot be correlated with exactly localised cerebral lesions. Critchley (1951) has criticised the efforts of those who would try to localise the lesion precisely in all cases, and both the few autopsies in the present series, and a study of numerous cases in the liter-

ature, afford evidence in favour of his views.

He has rightly emphasised that what is of importance is to recognise symptoms and signs suggestive of parietal lesions. These may frequently have to be sought specifically. In the writer's opinion, they will be found more often than the literature to date suggests. The present series is too small to permit firm conclusions, but it is suggestive that the ratio of cases with "parietal" symptoms and signs to those of uncomplicated hemiplegia was 13 to 37, or rather over 1 to 3.

Critchley (loc.cit.) has pointed out that the exact tabulation of parietal syndromes, and their equation with disease in one hemisphere or both, dominant or subordinate, may be misleading, and at times out of keeping with pathological findings at autopsy. He has drawn attention to certain clinical generalisations as suggestive of parietal lesions, as follows:-

- (1) A relative unilateral neglect, without necessarily any gross motor, sensory, or visual disorder. Herein lie for consideration such phenomena as anosognosia, confabulatory explanation of an affected limb, autotopagnosia, the partial autotopagnosia of Gerstmann's syndrome, anosodiaphoria, and sensory inattention, to name the more important.
- (2) Disorders in the memory of, or in the conception of, spatial relationships, especially those in three dimensions - a defect of Spearman's "K" factor, or

the ability to obtain, manipulate and utilize spatial imagery.

Arising from (1) and (2) he would rank unawareness or neglect of extra-personal space.

(3) "Subtle defects in the highest level of sensory integration", in particular tactile inattention, and tactile, visual or auditory allachaesthesia. These might well be classified in Group 1, where sensory inattention has already been mentioned.

(4) Arising from defects of spatial appreciation or integration, "difficulties in two-dimensional and three-dimensional motor performances" - constructional apraxia, apraxia for dressing, and ideo-motor apraxia.

(5) "Handicaps in the domain of language", particularly on the receptive side, including certain forms of dysgraphia and agraphia (e.g., in Gerstmann's syndrome).

Ascribing such defects to impaired parietal function is no more than a method of expressing the role of the parietal lobes as sensory receiving areas, apperceptive, correlative and integrative areas, and the seat of memory and discriminating ability. By virtue of their proper function, the individual can relate himself both to his person and his environment, and having done so, he can set in motion the mechanisms for appropriate motor activity, should that be required, whether by bodily movement, speech or writing.

However, if we except patients who display dementia or gross clouding of consciousness, there is now ample evidence that anosognosia (for hemiplegia) and autotopagnosia arise from lesions of the thalamo-parietal projection or parietal cortex of the subordinate hemisphere; likewise, Gerstmann's syndrome and its variants follow lesions of the dominant parietal lobe. This is one of the few conditions where rather exact localisation is generally possible (although one patient in this series coming to autopsy proved an exception to this rule). When we turn to disorders of spatial perception, the difficulties are greater, in that such syndromes seem to occur usually as a result of bilateral parietal damage, more rarely from lesions of the subordinate hemisphere, but at times from non-parietal causes, e.g. frontal tumour.

Tactile inattention, although usually a sign of a parietal lesion, has occasionally, though rarely, been found in association with high spinal lesions. Various other symptoms and signs, in particular agnosias, aphasias and apraxias, may stem from parietal lesions; they have not been included for study in the present instance in that, apart from the modified forms of aphasia or apraxia sometimes encountered in Gerstmann's syndrome, and the varieties of visual-spatial agnosia responsible for disorders of spatial appreciation, they were not

specifically noted in this series of cases. Similarly, disorders of personality of the type ascribed to lesions of the frontal lobes are in fact quite often the result of parietal lesions (Blake Pritchard (1952)). They likewise were not encountered, and are not further considered.

What must be ensured is not only that evidence of parietal dysfunction should be sought with care, but that our methods of examination are critical, and themselves beyond criticism. In the author's opinion, for example, acalculia may be assumed to represent a partial Gerstmann syndrome, when in fact it may actually indicate mental retardation or defective education. Similarly, many people in full possession of their faculties have difficulty in carrying out the simplest drawings, and there is in normal people a wide range of ability to think (and act or draw) in terms of plans or maps. We must not only be constantly critical of our methods of examination, but we must interpret the results with due regard to the state of education of our patients, and with proper knowledge of the ranges of normal. It seems reasonable, for example, to treat a patient's ability to draw a map with circumspection. But if one side of it be completed with care, while the other is almost wholly neglected; or if, as occurred in the instance of Case 3, the towns are placed in the sea, and there is hopeless confusion between the different points of the compass, then we may justly

be suspicious of parietal dysfunction.

Confusion between incapacity due to paresis and genuine apraxia has, of course, long been a pitfall for the unwary. And we must remind ourselves on occasion of the possibility that apparent defects in gnosis are in fact symptomatic of imperfect ability to express ideas, and therefore really forms of dysphasia (which itself may or may not suggest a parietal lesion).

A suggested scheme of examination is presented, as follows:-

- (1) What is the patient's state of consciousness?
How well oriented is he?
- (2) Is he right-handed or left-handed? Is there a family history of left-handedness?
- (3) Does he realise the nature of his disability, or has he anosognosia?
- (4) Is there evidence of anosodiaphoria?
- (5) Is autotopagnosia present, and if so, of what variety is it - has the patient merely periodic amnesia for part of the body, or has it disappeared from consciousness, or is there a state of feeling of absence of part of the body?
- (6) Is there evidence of Gerstmann's syndrome in whole or in part? If a defect of finger appreciation is present, what is its nature - agnostic, aphasic, or apraxic?
- (7) Are there isolated forms of sensory dysphasia or aphasia?

- (8) Are there isolated forms of agnosia?
- (9) Are there isolated forms of apraxia?
- (10) Is there evidence of Parietal forms of sensory disturbance e.g. astereognosis, impaired two-point discrimination, faulty estimation of weights, volumes and shapes of objects?
- (11) Does the patient have sensory inattention, bearing in mind that hypaesthesia or homonymous hemianopia may confuse the issue? Is there allachæsthesia?
- (12) What is the patient's relationship to extra-personal space? Does he neglect it? Can he find a route properly? Are defects in route-finding consistent (i.e., does a tendency to turn always to one side, and to neglect the other, exist)? Can he describe routes and places, especially houses and streets, properly? Can he draw plans, and copy them with reasonable accuracy? Is there apparent neglect of one half of the plan? What is the patient's capacity for avoiding obstacles (assuming there are no gross visual handicaps)?
- (13) What are the patient's drawing and copying abilities? Can he with reasonable accuracy bisect a line or indicate the centre of a circle? Can he distinguish the relative lengths of lines? What is his capacity for judging distances (assuming he is possessed of binocular vision)? Can he copy simple patterns, e.g. letters, in two dimensions, and others e.g. with matches or Koh's blocks, in three dimensions?

(14) Has he visual agnosia (testing each half-field of vision separately) ? Has he apraxia for dressing?

As Critchley (1951) has said "But one most striking clinical feature of the patient with a parietal lesion is the variability of his performance. He may make a gross error one moment, and give a successful response the next. ----- We must not gloss over inconsistencies or record what we imagine the patient should have done or said. These very irregularities, variabilities, repetitions, hesitancies, changes of mind, erasures, and so on, are, I submit, important. ----- We can identify in these paradoxes and incongruities a number of fundamental disturbances, such as, for example, fluctuation of attention, ideational inertia, perseveration, in the the evocation of an easy task to the exclusion of one which is more difficult; lack of demarcation between figure and background; loss of simultaneous function or an inability to cope simultaneously with two tasks, concepts, or percepts; lack of inhibition of associated ideas; the use of cerebral by-passes. All these are phenomena which have been thoroughly described and studied by the Gestaltists, and by the organismic school of neurologists - Lange, Hughlings Jackson, Pick, Head and Goldstein, who have based themselves upon the philosophies of Herbert Spencer and of Bergson.

Such phenomena are stumbling-blocks in the pathway of those who would nail their tattered banners to the masthead of a rigid cerebral localization. They

are additional arguments against the existence within the brain of a hard and fast localization of function - as opposed to a certain specialization of function." With such views one can be in entire agreement.

Further advances in our knowledge of the effects of disordered parietal function will doubtless depend on a better understanding of neuro-physiology, and on developments in psychology, if at the same time a much-needed respect for philosophy, and in particular for logic, is regained by physiologists and psychologists alike. It seemed at one time that an attempt might be made to explain all the difficult problems of the body-image, of man's topographical relationship to his outer environment, of gnosis, praxis, and speech, along mechanistic lines based on cybernetics. But while the value of the study of cybernetics may be undoubted, the pseudo-scientific "philosophy" of many exponents of the subject is so puerile that it would deserve to disappear without further comment, were it not for the fact that it has been acclaimed so widely and so uncritically. It has been devastatingly condemned by Walshe (1952), who has additionally pointed out the folly of comparing the mind to an elaborate form of calculating machine, which is, as the product of mind, inevitably less than mind itself, and which, though capable of solving given problems, is devoid of the capacity for original thought.

As emphasised earlier, the present study has not the value it might have possessed had all cases ended fatally, and all been subjected to post-mortem examination. But it is presented with the object of drawing attention to the relatively common occurrence of "parietal" symptoms and signs, a matter which as yet appears not to be widely recognised. While the claims for exact localisation of lesions in all, or even the great majority of cases of parietal syndromes can no longer be upheld, it appears to the writer that a more thorough search for the presence of "parietal" symptoms and signs in appropriate cases, and a fuller appreciation of such clinical findings when they occur, is no mere academic exercise. Modern techniques such as air-studies, cerebral angiography and electro-encephalography, though often of the greatest value, must be used in addition to full clinical studies, not in place of them.

Summary.

It is suggested that symptoms and signs of parietal lobe dysfunction are commoner than has hitherto been supposed. Such a contention is based on the clinical study of a series of patients who were seen during the years 1948 - 1951. The frequency of such cases is compared with that of cases of hemiplegia without parietal symptoms and signs. The ratio of patients with evidence of parietal lobe disorders to those without was approximately 1 to 3. The main

syndromes encountered have been discussed, and a review of the extended meanings of certain syndromes has been made. The localising value of the main syndromes has been considered; it has been thought to be approximate only. A plea is made for the abandonment of so-called "exact localisation" of lesions on the basis of certain "parietal" symptoms and signs. But the desirability of examining carefully for the presence of such symptoms and signs is urged, as is the necessity of using critically tests for disordered parietal function.

Acknowledgement.

I wish to tender my sincere thanks to Dr.W.I.Card, under whose care the patients were, for allowing me to study them in detail.

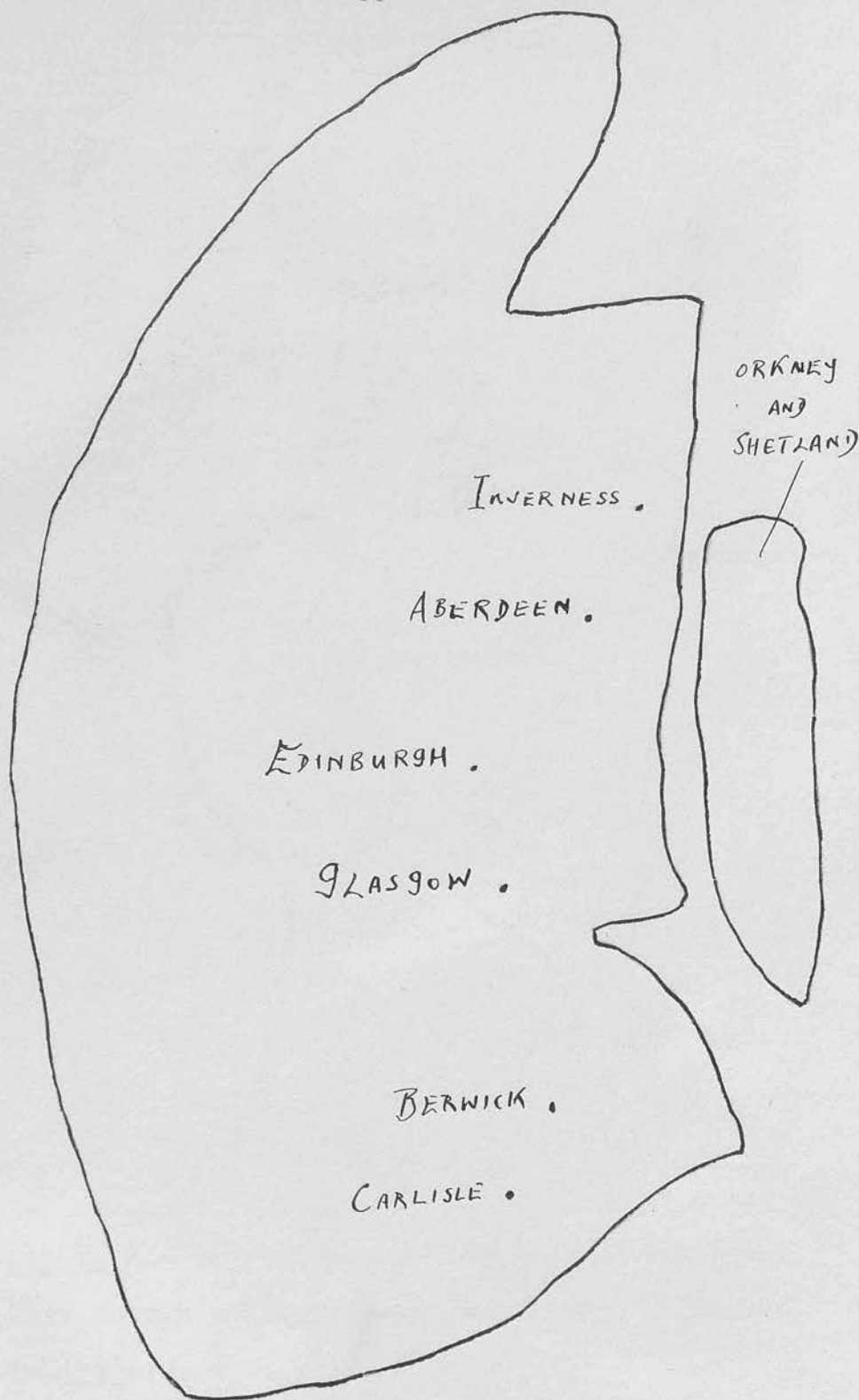


Figure 1:- Copy of Map of Scotland (Patient 3).

Note west coast, position of cities, and Orkney and Shetland.



Figure 2:- Copy of Map of Scotland (Patient 3).
Note poorly completed west coast, and towns set
in the sea.

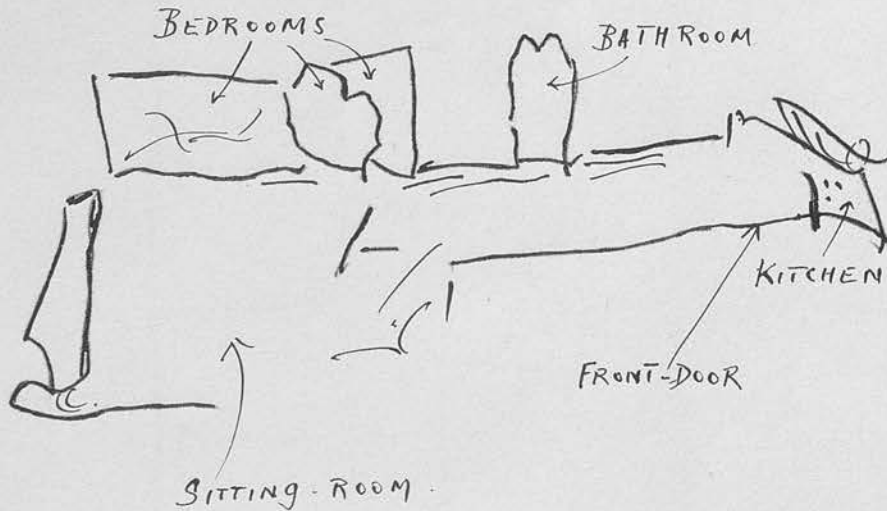


Figure 3:- Plan of house (Patient 4). This was made shortly after admission. Note the poor organisation of the whole. There is no evidence of neglect of one side, or of top or bottom.

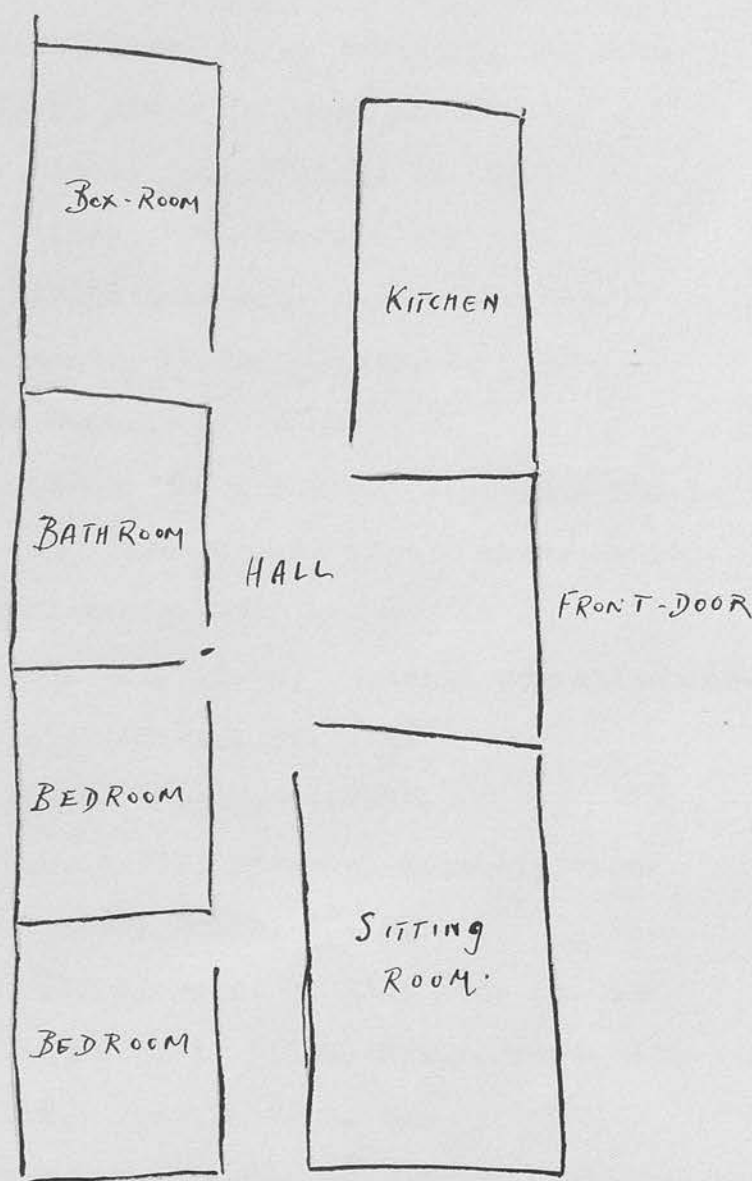


Figure 4:- Plan of house (Patient 4). This was made shortly before the patient was discharged from hospital. Although imperfect, it shows considerable improvement when compared with Figure 3.

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